

DOCUMENT RESUME

ED 345 401

EC 301 161

AUTHOR Silver, Archie A.
TITLE The Heterogeneity of ADHD and Some Implications for Education.
PUB DATE Mar 92
NOTE 21p.; Paper presented at the International Conference of the Learning Disabilities Association (Atlanta, GA, March 4-7, 1992).
PUB TYPE Speeches/Conference Papers (150)
EDRS PRICE MF01/PC01 Plus Postage.
DESCRIPTORS *Attention Deficit Disorders; Behavior Change; *Drug Therapy; Elementary Secondary Education; Etiology; *Hyperactivity; Individualized Programs; Interdisciplinary Approach; *Intervention; Learning Disabilities; Medical Evaluation; Needs Assessment; Outcomes of Treatment; Research Methodology; Special Education
IDENTIFIERS Methylphenidate

ABSTRACT

This paper examines factors possibly responsible for the poor outcomes of many children with attention deficit hyperactivity disorder (ADHD) and suggests possible ways of improving services to this population. It is concluded that each of the etiological factors identified for ADHD has its own characteristics, driven by genetic vulnerabilities and shaped by the child's life experiences. Specific ADHD is seen to involve inattention, impulsivity, and hyperactivity without any etiological implication. Specific ADHD is subdivided into two major groups, the behavioral subtype and the cognitive subtype, which respond to different forms of intervention including stimulant medication. The failure to account for heterogeneity in the ADHD population is seen to be responsible for the mixed findings of studies on the effectiveness of methylphenidate therapy. Schools are encouraged to recognize the heterogeneity of the ADHD syndrome; provide comprehensive diagnosis in cooperation with medical and other professionals; and devise individually appropriate combinations of remedial education, medication, behavioral modification, and support. (31 references)

* Reproductions supplied by EDRS are the best that can be made *
* from the original document. *

ED345401

The Heterogeneity of ADHD and Some Implications for Education*

Archie A. Silver, M.D.**

U.S. DEPARTMENT OF EDUCATION
Office of Educational Research and Improvement
EDUCATIONAL RESOURCES INFORMATION
CENTER (ERIC)

☒ This document has been reproduced as
received from the person or organization
originating it.
☐ Minor changes have been made to improve
reproduction quality.

• Points of view or opinions stated in this docu-
ment do not necessarily represent official
OERI position or policy.

"PERMISSION TO REPRODUCE THIS
MATERIAL HAS BEEN GRANTED BY

Archie A. Silver

TO THE EDUCATIONAL RESOURCES
INFORMATION CENTER (ERIC)."

The long term outcome in terms of social adjustment and educational achievement of children identified as having a hyperactive disorder is, in general, poor (G. Weiss & Hechtman, 1986; Loney, Whaley-Klatin, Kosier and Comboy, 1983; Borland and Heckman, 1976). In a more recent prospective follow-up study (Mannuzza *et al*, 1991) 50 boys diagnosed as hyperactive in childhood and followed 6-14 years into adolescence, were compared with their siblings and with controls, so that 50 subject-sibling pairs were studied along with 50 adolescents matched for age and social-class. While the prevalence of antisocial behavior (28%) did not significantly discriminate probands from their brothers (18%), the probands with antisocial disorder "had a more severe form of syndrome than their brothers ... all probands with an ongoing antisocial behavior had been suspended or expelled from school, had gotten into trouble for failing to conform to school rules, were repeatedly truant, had demonstrated reckless, dangerous behavior ... and had ignored rules that were set at home" (p.749). Focusing only on educational achievement, up to 50% of children diagnosed as attention deficit hyperactivity disorder may have been retained in grade (Barkley 1990), 40% have received special educational services (Barkley, 1991) and 35% may have failed to finish high school (Weiss & Hechtman, 1986). It is clear that our present medical,

* Read at the International Conference, LDA, Atlanta, GA, March 4-7, 1992.

** Professor of Psychiatry, Director of Child and Adolescent Psychiatry, University of South Florida Medical School, Tampa, Florida.

The author appreciates the literature review of Samantha L. Liu, a fourth year medical student at the University of South Florida, the literature searches conducted by our librarian, Ms. Marilyn Wilson, and the critical review of this manuscript by Dr. Rosa Hagin.

EC 301161

psychological and educational approaches to these children are not adequately meeting their needs.

Current Federal Policy (Davila, Williams & McDonald, 1991) now places the task of providing special educational and related services for the ADHD child who meet criteria under part B of the Individuals with Disabilities Education Act, or as a handicapped person under Section 504 of the Rehabilitation Act of 1973, squarely on the responsibility of state and local educational agencies. It seems appropriate, therefore, to review some of the factors which may be responsible for the unhappy outcome of many children with ADHD and to suggest possible ways of improving services for them.

Of first importance in providing appropriate services for these children is the recognition that the diagnosis of ADHD in accordance with the criteria of DSM-III-R (1988) yields a heterogeneous group of children that may differ in clinical manifestations, etiology, natural history, management, and prognosis. The philosophy of DSM-III-R is said to be athoretical. It does not claim to classify groups on the basis of etiology, but it simply offers a cluster of symptoms which appear to be related; in case of ADHD, difficulty with sustained attention, impulsivity and hyperactivity. The criteria for this diagnosis are satisfied if at least 8 of 14 symptoms proscribed by DSM-III-R, have been present for at least 6 months, are more than expected as compared with children of the same mental age and have had their onset before the age of 7. DSM-III-R makes no pretense of defining ADHD as a homogeneous etiological group. Unfortunately DSM taxonomy takes on a life of its own and the symptoms of attention defect, impulsivity, and hyperactivity soon are treated as though they comprise a discrete disorder. Stimulant medication is then almost reflexly given as

though that would cure all the problems in the child with the symptom cluster of ADHD. As Ostrom & Jenson (1988) point out, the ADHD label, if taken literally, has limited meaning "it lacks a clearly conceptualized research base and clearly articulated definitions of the primary components of attentional defect, impulsivity, and hyperactivity" (p. 263) "ADHD may include a more loosely defined set of common childhood behavior problems: non-compliance, academic difficulties, social skills defects, aggression, over-activity and attention deficits" (p.264). Even the issue of whether attention deficit without hyperactivity and attention deficit with hyperactivity belong in the same taxonomic grouping is open to question. In DSM-III the two forms of ADD are considered separately; DSM-III-R combines them into a unitary, supposedly equivalent, disorder. A review of the literature from 1980 to 1989, however, (Driscoll & Zecker, 1991) finds ADHD children to be different from ADD/wo children. ADHD children are impulsive, distractible, active, aggressive with conduct problems while the ADD children without hyperactivity are generally shy, withdrawn, sluggish, frequently repeat grades and are more anxious and depressed than ADHD children. They may represent two different disorders. In management the impulsive, aggressive child will require a more structured, distraction free approach than the encouraging, supportive approach needed by the shy, withdrawn child.

Equally important are the etiological factors obscured by the diagnosis of ADHD, etiological factors in which attentional problems, impulsivity and hyperactivity may be only a part of the clinical presentation, etiological factors which require different approaches to management and may offer different natural histories and prognosis. A list of cause of sustained attention deficits and hyperactivity, taken from Silver and Hagin (1990) is seen in Table I. ADHD may arise from many causes, ranging from

being temperamentally impulsive (Thomas and Chess, 1980) to severe psychiatric disorders and, of course, any combination of etiological factors may act together. To call each of these ADHD is to mistake a symptom for a disease.

Michael, for example, was 10 years of age, one of fraternal twins in a high income professional family, in the 4th grade of a parochial school when he was referred to our clinic because of his restless, disruptive behavior, his quick temper, and his resistance to do any written work. In kindergarten, he had been diagnosed as attention deficit disorder with hyperactivity and had been treated with methylphenidate, which only made him more irritable and demanding. Psychological testing at that time (age 6 years, 2 months) found Michael's intellectual functioning on the WISC-R in the low average range (verbal, average; performance, low average) with weakness noted in the Picture Completion, Information, Similarities and Comprehension sub-tests. On our examination, Michael was a short, stocky, dark-haired, freckle-faced boy in contrast to his taller, fair-haired twin. He was taciturn, sulky, and demanding but soon the anxiety underlying his behavior emerged: he is afraid to go into his swimming pool for fear of a shark, he fears going to sleep at night because he thinks "Jaws" is on the carpet in his room, he thinks a snake is in his closet, and he surrounds his bed with tall furniture so that nothing can get by. At night, he checks the closet, looks under the bed, makes sure that there is nothing on the ceiling and, even then, needs to have his dog sleep in his room. He dreams of a monster chasing him. He hears God talking to him telling him not to be afraid. He feels he can get whatever toys he wants by just being bad. At school, he resents any written work and knows that if he fails his teachers will give him an easier test until he passes. He wishes to be the strongest boy

in his class. He blames his problems on his parents, his brother, and his teachers and he does not like his classmates either.

On physical examination Michael was a clumsy child with awkward gross-motor coordination and poor fine-motor coordination; the left side of his mouth drooped. His ocular pupils were eccentric and there was a sustained nystagmus on lateral gaze. Synkinesis was marked. Body image was markedly impaired in right-left discrimination, finger-gnosis, and in constructive finger praxis. Perceptually, he had difficulty with the visual recall of asymmetric figures, his visual-motor function (Bender-Gestalt) was at a 7-1/2 year old level (less than 5th percentile) with errors of distortion, verticalization, and incompleteness. In addition, there were severe immaturities in auditory perception, particularly in temporal sequencing, and in immediate auditory recall. On psychometric examination at age 10 years, Michael earned a Full Scale I.Q. (WISC-R) of 90 (Verbal-91, Performance-91) with sub-test scatter particularly in the performance scale with scaled score of 7 on Block Design, 6 on Object Assembly, 7 on Picture Completion, in contrast to 12 on Picture Arrangement, and 12 on Coding. On the Detroit Test of Learning Aptitude there were marked deficits in visual-motor tasks, and in auditory memory with auditory attention span for related syllables attaining but a 5-6 year level. As demands became more complex, Michael became more irritable, more restless and less attentive. His history revealed that labor with the twins was induced. Michael, the first born, required resuscitation. EEG is reported as dysrhythmic grade II with diffuse high voltage slow waves.

Michael represents a child who, although fitting the criteria for attention deficit hyperactivity disorder, has much more than that. He has a diffuse static encephalopathy, possibly occurring at birth, which has caused scattered central processing defects, a vulnerability to anxiety in addition to the impulsivity and attention deficit which was apparent as he entered kindergarten.

There are many Michaels in the classroom, as many as 5% of the average, mainstream first grade (Hagin, Beecher & A. Silver, 1982), perhaps one in each classroom of 25 children. To provide appropriate and adequate management for children with subtle organic central nervous system defects as well as for all the other children who may be suffering from any combination of problems listed in Table I, a thorough comprehensive examination, involving medical, psychological, educational, and social parameters is a minimal requirement. The cooperative efforts of many disciplines is needed.

Subtle organic structural defects of the central nervous system as illustrated by Michael, represent only one of the many factors in which attention deficit-hyperactivity may be a prominent symptom (Table I). The association of ADHD with conduct disorder, depression, anxiety, Tourette's syndrome, developmental disorders has been documented and has been termed co-morbidity. This raises a number of questions. Are these disorders discrete and independent disease entities in which attention, impulsivity and hyperactivity are symptoms just as fever is a symptom of many different disorders? Are they more homogeneous subgroups of attention deficit disorders or the expression of phenotypic variability of the same disorder? Does the

development of one syndrome increase the risk for co-morbid disorders? Do the co-morbid disorders and ADHD share common familial vulnerabilities?

A review of the literature on disorders frequently co-existing with ADHD by Biederman, Newcorn and Sprich (1991) reported 30-50% of children with ADHD also may be diagnosed as conduct disorder, 35% with oppositional defiant disorder, 15 to 75% with mood disorders, 25% with anxiety disorders. Sixty percent of children with Tourette's syndrome have by DSM-III-R definition, an attention deficit disorder. The relationship of ADHD with learning disorders will be considered later in this paper. Recognizing again the athoretical taxonomy of DSM-III and DSM-III-R, categorical answers to the questions raised by co-morbid disorders, which are really combination of symptoms which frequently appear together, cannot at this point be given. For example, there is evidence that ADHD and conduct disorder are manifestations of the same underlying, as yet unknown, pathology. On the other hand, there is also evidence that ADHD and conduct disorder are independent entities with little correlation between hyperactivity and aggression. Conduct disorder, however, was significantly correlated with aggressive behavior and delinquency in adolescence (Szatmari, Boyle and Offord, 1989) suggesting that the symptoms may share a common origin. Even the relationship between ADHD and Tourette's syndrome is not understood. One of the earliest manifestations of Tourette's syndrome is attention deficit and hyperactivity. Comings and Comings (1988) believe that there is a genetic relationship between ADHD and Tourette's syndrome and that a Tourette's syndrome gene accounts for approximately 10-30% of ADHD. Pauls, Hurst, Kruger *et al* (1986), however, do not find evidence for this at all. The exacerbation of the motor

and vocal tics by the very stimulant drugs which obtund the symptoms of ADHD add further complexity to the relationship between the two syndromes.

What one may conclude from a review of all the possible causes of inattention, impulsivity and hyperactivity, is that ADHD is far from a simple set of symptoms, arising from a single source and requiring the same treatment. Each of the etiological factors listed in Table I has its own characteristics, driven by genetic vulnerabilities and shaped by the experiences of the child's life. As indicated with the case of Michael, psychiatric, neurological, psychological, family and educational parameters must be explored to understand as best we can, the parameters of the child's problems. The Federal mandate placing ADHD in the category of the handicapped child imposes the responsibility for this type of comprehensive evaluation on the school system.

Even when such an evaluation is provided, however, and the etiological factors and signs underlying the symptoms of inattention, impulsivity, and hyperactivity revealed, there remains a core of children for whom no etiology can be found. These may be the "pure" ADHD children. Analogous to the term specific language disability, we may say the "pure" ADHD child is suffering from a specific attention deficit disorder; specific in the medical sense that it does not have etiological implication, which at this point is a matter of speculation and in the general sense that it focuses on a major cluster of signs: inattention, impulsivity, and hyperactivity. Of course this group, too, may be heterogeneous but for practical and research purposes it, at least, identifies one group of children with ADHD, supposedly uncontaminated by the etiological factors listed in Table I. Within this group of pure or specific ADHD children, two subgroups have been described (August and Garfinkel, 1989; Silver and

Hagin, 1990) -- the first a so-called behavioral subtype characterized by the pathognomonic triad, may be described on a "continuum of severity with the most severe showing behavioral features indistinguishable from conduct disorders" (August and Garfinkel, p.739). The second group, a cognitive subtype is characterized not only by the pathognomonic triad but also, and most importantly, by central nervous system processing deficits which are indistinguishable from those of specific learning disabilities; namely, problems with perceptual tasks of spatial orientation and/or temporal organization with all the subsequent deficits in hierarchical functions which arise from these basic processing defects.

The recognition of these two subgroups of specific attention deficit hyperactivity disorder, behavioral and cognitive, raises important issues in understanding the relationship of ADHD to learning and to subsequent management of these children. The educational implications of these sub-groups have been described in A. A. Silver & Hagin, 1990. It has been reported that as many as 94% of children with ADHD have a "learning disability" (L.B. Silver, 1981). To understand the implications of such a statistic, definitions of learning disability are most important here. If we define a specific learning disability as a condition involving specific central nervous system processing deficits then the majority of the children with ADHD do not have such deficits. In August and Garfinkel's experience (1989) and Sprich (1991), 80% of children with ADHD do not have such processing deficits and the figure of 94% is misleading. Twenty percent of them do, however, and thus, approximately 1 of 5 children with ADHD will also have a specific learning disability. This does not mean, however, that the remaining 4 of 5 children with ADHD do not have learning problems. The deficits in attention and the impulsivity of the child with "pure" ADHD may, in

themselves, prevent a child from being able to sustain focus on a specific task and to maintain concentration to complete it. Thus, the ADHD child who does not have central processing defects, may indeed have a learning problem. This problem, which we define as a learning disorder in the sense that his academic achievement is below expectancy for his I.Q. and educational experience, is different from that of the child with a specific learning disability who may also be impulsive and inattentive. The difference is readily apparent in two ways: clinically, the linguistic problems in temporal organization and/or spatial orientation of the SLD child are not found in the pure ADHD, and most important in management where the processing defects of the SLD child do not respond to the stimulant medication which will control the impulsivity and attention problems of ADHD. In the child with ADHD without central processing deficits, the problems in attention and impulsivity may be the limiting factor in learning, while in the child with problems in spatial orientation and temporal organization, attention and impulsivity is only one factor in limiting academic learning. For a child with central processing defects, these defects will not respond to stimulant medication and the treatment of the SLD itself is an essential part of management. Indeed, in our experience for many children with SLD with ADHD remedial teaching, without the use of stimulant drugs, may be sufficient in itself to sustain attention and improve learning.

A review of the literature substantiates these conclusions. For the most part, however, literature does not make the distinction between pure ADHD and other etiological factors that may cause the symptoms of attention deficit, impulsivity and hyperactivity. The sample of children under study, therefore, may be heterogeneous and the outcomes contaminated by the heterogeneity of the sample. This problem is, of

course, similar to that encountered in many studies of learning disabilities. Nevertheless, the literature is in general agreement that while in the short term (exact limits unknown) stimulant drugs do improve laboratory tests of sustained attention, this is not matched by evidence of improved academic learning (Barkley and Cunningham, 1978, Barkley, 1977, Gittleman-Klein & Klein, 1976, Gittleman, Klein and Feingold, 1983, Douglass, 1983, Aman & Werry, 1982). Aman and Werry conclude that stimulant drug related changes in ADHD "occurred mostly in areas unrelated to basic cognitive deficits" (p. 36) and that "while most investigations can be faulted on diagnostic heterogeneity, dose, or direction of medication, it seems reasonable to conclude that at present, stimulants have no clear role to play in (improving) learning disabilities" (p. 37). Further, Sprague and Sleator (1977) have reported that in a group of ADHD children, the error rate on a continuous performance test which is improved on low doses (10-20- mgm/day) of methylphenidate, progressively deteriorates with higher doses.

In contrast to the conclusions of our own experience and of the literature cited in the preceding section, however, there has been criticism that "methodological deficiencies may account for failure ... to obtain consistent effects on achievement. First, drug dose was not systematically varied ... second, standardized achievement tests used lack the sensitivity to detect short-term effects. Third in most studies no effort was made to control academic intervention" (Richardson, Kapietz, Winsberg, Matinsky and Mandel (1988, p. 79). In the Richardson *et al* study, 43 children with ADHD diagnosed also as Developmental Learning Disorder if they met DSM-III criteria for DLD and who achieved no higher than 75% of expected academic achievement relative to their grade placement, were divided into 4 groups, each of which received a different dose of

methylphenidate (placebo, 0.3 mgm kilo/day, 0.5 mgm and 0.7 mgm) and received a program of reading instruction, the Integrated Skills Method (Richardson, 1984) over a 7 month period, in two weeks of which (the 15th and the 28th week) all children were placed on placebo and academic tests administered. In the first half of this study, the higher the level of methylphenidate in plasma the better the reading gain.

However, Richardson *et al*, conclude that "to the degree that methylphenidate is effective in controlling ADHD symptoms improvement in reading achievement can be expected" (p. 85). Our problem with this study is that there is no evidence that the children classified as learning disorders were really children with central processing defects of SLD and that while the children do improve as a function of improved attention and concentration, there is no evidence that the central processing defects are corrected. A series of investigations by Pelham and his associates (Pelham, 1985; Pelham, Bender, Caddell, Booth & Moorer, 1985, Pelham & Murphy, 1985, Carlson, Pelham, Swanson & Wagner, 1991) conclude that methylphenidate improves the arithmetic performance of ADHD children; children solve problems more quickly and accurately and move from problem to problem faster when receiving methylphenidate than when receiving placebo. Unfortunately, these reports, too, do not document the neuropsychological defects as well as the ADHD symptoms of their subjects.

Our conclusion is in no way to minimize the contribution of stimulant drugs in the management of ADHD. On the contrary, most children, approximately 75%, will respond to methylphenidate in reducing overactive and impulsive behaviors and in improving laboratory tests requiring sustained attention. However, stimulant drugs are not miracle drugs, they cannot do all things for all children. Stimulant drugs are not the cure for the central processing defects which may appear in approximately

20% of children with ADHD. In addition, in our experience, the child who fails to respond to stimulant medication and to other drugs currently in use for the ADHD syndrome (i.e.: tricyclic antidepressants, clonidine) is suffering from more than pure or specific attention deficit disorder, and has in addition some combination of complications outlined in Table I.

Thus, the generally poor adjustment and educational outcome of children with a diagnosis of Attention Deficit Hyperactivity Disorder may, in part, be to our failure to recognize the heterogeneity of that disorder; that, in reality, the diagnosis of ADHD in accordance with DSM-III-R offers a cluster of symptoms namely difficulty with sustained attention, impulsivity and hyperactivity; that there are many causes and combination of causes for these symptoms, each of which may differ in clinical manifestations, natural history, management and prognosis. Even when we can isolate a child with the core symptoms of inattention, impulsivity and hyperactivity, without any of the complicating etiological factors, the so-called "pure ADHD", these children, too, are further dichotomized into those with and those without central processing defects.

The importance of these findings for education is that the educational system cannot reflexly place the hyperactive, inattentive child into a class for emotionally disturbed children. This is just as bad as the reflex administration of stimulant drugs to children who are overactive. Schools need to recognize the heterogeneity of the ADHD syndrome, provide comprehensive diagnosis without undue delay, and with that information as a background, devise appropriate and adequate class placement and management. This recommendation is, itself, complex. It implies a close, working

relationship among a multiprofessional team: medical people to provide their component of the comprehensive evaluation and to prescribe and monitor medication, as needed; family therapists to work with the environmental factors shaping behavior; counsellors to provide individual support; special education classes to improve the central processing skills which will be found in one of every five children with ADHD; and, trained classroom teachers to understand and cope with inattention and restlessness. The child with Tourette's syndrome or with hearing impairment, the SLD child, the child with visual-motor problems, the retarded child, the child with little environmental support, and disturbed object relations, all may present the same ADHD but all require different combinations of remedial education, medication, behavioral modification and support.

It now becomes the legal mandate for the school system to supply these individual needs.

Table 1 *Causes of Sustained Attention Deficits and Hyperactivity (not in order of prevalence)*

1. Task too boring or too difficult
 2. Specific language disability
 3. Anxiety
 4. "Conduct disorder" (DSM-III-R)
 5. Structural defect of the central nervous system
 6. Cognitive retardation
 7. Depression; manic-depression
 8. Inappropriate or inadequate early object relations
 9. Severe developmental disorders (severe language lags, autism, pervasive developmental retardation)
 10. Schizophrenia
 11. "Attention deficit hyperactivity disorder" (DSM-III-R)
 12. Temperamental impulsiveness
 13. Tourette's syndrome
 14. Toxic substances (i. e., lead)
 15. Hearing impairment
-

*Table 13-2 from: A.A. Silver and R.A. Hagin (1990),
Disorders of Learning in Childhood, J. Wiley, New York.*

REFERENCES

- Aman, M. & Werry, J. (1982). Methylphenidate and diazepam in severe reading retardation. *J. Am. Acad. Chi. Psi.*, 21, 31-37.
- August, G. J. and Garfinkel, B.D. (1989). Behavioral and cognitive subtypes of ADHD. *J. Am. Acad. Child & Adolesc. Psychiatry*, 28, 739-748.
- Barkley, R.A. (1977). A review of stimulant drug research with hyperactive children. *J. Child Psychol. & Psychiatry*, 18, 137-165.
- Barkley, R.A. and Cunningham, C.E. (1978). Do stimulant drugs improve the academic performance of hyperkinetic children? *Clinical Pediatrics*, 17, 85-92.
- Barkley, R.A., Anastopoulos, A.D., Guevremont, D.C., and Fletcher, K.E. (1991). Adolescents with ADHD: Patterns of behavioral adjustment, academic functioning and treatment utilization. *J. Am. Acad. Child Adolesc. Psychiatry*, 30, 752-761.
- Barkley, R.A., Fischer, M., Edelbrock, C., and Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria I: An 8 year prospective follow-up study. *J. Am. Acad. Child Adolesc. Psychiatry*, 29, 546-557.

Biederman, J., Newcorn, J. and Sprich, S. (1991). Co-morbidity of attention deficit hyperactivity disorder with conduct, depressive, anxiety and other disorders. *Am. J. Psychiatry*, 148, 564-577.

Borland, B.L. and Heckman, H.K. (1976). Hyperactive boys and their brothers: a 25 year follow-up study. *Arch. Gen. Psychiatry*, 33, 669-675.

Carlson, C.L., Pelham, W.B., Jr., Swanson, J. M, & Wagner, J.L. (1991). A divided attention analysis of the effects of methylphenidate on the arithmetic performance of children with attention deficit disorder. *J. Child Psychol. & Psychiatry*, 32, 463-471.

Comings, D.E. and Comings, B.G. (1988). Tourette's syndrome and attention deficit disorder. In D. J. Cohen, R. D. Bruun, & J.F. Leckman (Eds.), Tourette's Syndrome and Tic Disorder, pp. 119-136. New York: Wiley.

Davilla, R.R., Williams, M.L., and MacDonald, J.T. (1991). Clarification of policy to address the needs of children with Attention Deficit Disorders within general and/or special education. Memo to Chief State School Officers; Washington, D.C., U.S. Department of Education.

Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, revised (1988). Washington D.C.: American Psychiatric Association.

- Douglas, V.I. (1983). Attentional and cognitive problems. In M. Rutter (Ed.), Developmental Neuropsychology, pp. 280-329. New York: Guilford.
- Driscoll, M.S. and Zecker, S.G. (1991). Attention Deficit Disorder: Are there subtypes? A review of the literature from 1980 - 1989. *Learning Disabilities*, 2, 55-64.
- Gittelman, R., Klein, D.F. and Feingold, I. (1983). Children with reading disorders -- II. Effects of methylphenidate in combination with reading remediation. *J Child Psychol. & Psychiatry*, 24, 193-212.
- Gittelman-Klein, R. and Klein, D.F. (1976). Methylphenidate effects on learning disabilities. *Arch. Gen. Psychiatry*, 33, 655-664.
- Hagin, R.A., Beecher, R., and Silver, A.A. (1982). Definition of learning disabilities: A clinical approach. In J.P. Das, R.F. Mulcahy and A.E. Wall (Eds.) Theory and Research in Learning Disabilities, pp. 45-57. New York: Plenum.
- Loney, J., Whaley-Klatin, M.A., Kosier, T. and Conboy, J. (1983). Hyperactive boys and their brothers at 21: Predictors of aggressive and antisocial outcomes. In K.T. VanDusen and S.A. Mednik (Eds.), Prospective Studies of Crime and Delinquency, pp. 181-206. Boston: Kluwer-Nighoof.

- Mannuzza, S., Gittelman-Klahn, R. and Addalli, K.A. (1991). Young adult mental status of hyperactive boys and their brothers: A prospective follow-up study. *J. Am. Acad. Child Adolesc. Psychiatry*, 30, 743-751.
- Ostrom, N.N. and Jenson, W.R. (1988). Assessment of attention deficits in children, *Professional School Psychology*, 3, 254-269.
- Pauls, D., Hurst, C.R., Kruger, S.D. *et al* (1986). Gilles de la Tourette's syndrome and attention deficit disorder with hyperactivity: evidence against a genetic relationship. *Arch. Gen. Psychiatry*, 43, 1177-1179.
- Pelham, W.E. (1985). Effects of psychostimulant drugs on learning and academic achievement of children with attention deficit disorders and learning disorders. In J.K. Torgesen and B. Wong (Eds.), Psychological and Educational Perspectives on Learning Disorders. Orlando, FL: Academic Press.
- Pelham, W.E. and Murphy, A.A. (1985). Behavioral and pharmacological Rx of attention deficit disorders and conduct disorders. In M. Hersen (Ed.), Pharmacological and Behavioral Rx: An Integrative Approach, New York: Wiley.
- Pelham, W.E., Bender, M.E., Coddell, J., Booth, S., and Moorer, S.H. (1985). Methylphenidate and children with attention deficit disorders: Dose effects on classroom, academic and social behavior. *Arch. Gen. Psychiatry*, 42, 948-952.

- Richardson, E., Kupietz, S., Winsberg, B., Maitinsky, S. and Mandell, N. (1988).
Effect of methylphenidate dosage in hyperactive reading disabled children: II
reading achievement; *J. Am. Acad. Child & Adolesc. Psychiatry*, 27, 78-87.
- Silver, A.A. and Hagin, R.A. (1990). Disorders of Learning in Childhood, New York:
Wiley.
- Silver, L.B. (1981). The relationship between learning disabilities, hyperactivity,
distractibility and behavioral problems. *J. Am. Acad. Child Psychiatry*, 20,
385-397.
- Sprague, R.L., and Sleator, E.K. (1977). Methylphenidate in hyperactive children:
Differences in dose effects on learning and social behavior. *Science*, 198,
1274-1276.
- Szatmari, P., Boyle, M., and Offord, D. R. (1989). ADHD and conduct disorder: degree
of diagnostic overlap and differences among correlates. *J. Am. Acad. Child
Adolesc. Psychiatry*, 28, 865-872.
- Thomas, A. and Chess, S. (1980). The Dynamics of Psychological Development. New
York: Brunner/Mazel.
- Weiss, G. and Hechtman, L. (1986). Hyperactive Children Grown Up. New York:
Guilford.